



UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/517,251	12/07/2004	Phillip Mark Hogarth	4602AL-I	2961

22442 7590 10/06/2006

SHERIDAN ROSS PC
1560 BROADWAY
SUITE 1200
DENVER, CO 80202

EXAMINER

WEHBE, ANNE MARIE SABRINA

ART UNIT	PAPER NUMBER
----------	--------------

1633

DATE MAILED: 10/06/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

10/517,251

Applicant(s)

HOGARTH ET AL.

Examiner

Anne Marie S. Wehbe

Art Unit

1633

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 24 July 2006.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-3,5 and 7-12 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-3,5 and 7-12 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☐ Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: _____

DETAILED ACTION

Applicant's amendment and response received on 7/24/06 has been entered. Claims 4, 6, and 13-42 are canceled. Claims 1-3, 5, and 7-12 are pending and under examination in the instant application. Those sections of Title 35, US code, not included in this action can be found in the previous office action.

Please note that the examiner of record and art unit for this application have changed. See the last page of this action.

All rejections of previously pending claim 4 have been withdrawn in view of the cancellation of this claim.

Drawings

The objection to the drawings as failing to comply with 37 CFR 1.84(p)(5) is withdrawn in view of applicant's amendment to the brief description of the drawings on page 7 of the specification.

Claim Rejections - 35 USC § 112

The following new rejection under 35 U.S.C. 112, first paragraph, for lack of written description applies. Since this new rejection is not necessitated by applicant's amendment, this

Art Unit: 1633

office action is non-final. Please note as well that the following written description and enablement rejections are separate rejections.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-3, 5, and 8-12 are newly rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The specification does not provide sufficient written description for the genus of rodent strains that are resistant to collagen-induced arthritis. It is noted that the order Rodentia includes around 1500 mammalian species. The specification does not describe or provide sufficient guidance for identifying any species of rodent other than an F1 C57Bl/6 X SJL mouse that is resistant to collagen-induced arthritis. The specification, while generally referring to non-human mammals and rodents, only teaches a single mouse strain with the claimed characteristic of resistance and does not teach or disclose any other species of rodents, such as squirrels, hamsters, prairie dogs or marmots. The specification further does not describe the particular structural, physical, or functional characteristics of the F1 C57Bl/6 X SJL mouse that renders it resistant to collagen-induced arthritis such that this would allow other rodents with similar characteristics to

Art Unit: 1633

be isolated or identified. As such, of the large numbers of rodent species encompassed by the claims, the specification only provides adequate written description for a single species, the F1 C57Bl/6 X SJL mouse. Since the specification fails to provide any description of any other rodent with the claimed characteristic of resistance, the skilled artisan cannot envision the detailed chemical structure of the encompassed rodents other than the F1 C57Bl/6 X SJL mouse which has the characteristic of resistance to collagen-induced arthritis.

The Revised Interim Guidelines state, “ when there is substantial variation within the genus, one must describe a sufficient variety of species to reflect the variation within the genusIn an unpredictable art, adequate written description of a genus which embraces widely variant species cannot be achieved by disclosing only one species within the genus” (Column 2, page 71436, or the Revised Interim Guidelines for Written Description). Further, *Vas-Cath Inc. v. Mahurkar*, 19 USPQ2d 1111, clearly states that "applicant must convey with reasonable clarity to those skilled the art that, as of the filing date sought, he or she was in possession of the invention. The invention is, for purposes of the 'written description' inquiry, whatever is claimed." (See page 1117). The instant specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See *Vas-Cath* at page 1116). An applicant shows possession of the claimed invention by describing the claimed invention with all of its limitations using such descriptive means as words, structures, figures, diagrams, and formulas that fully set forth the claimed invention. *Lockwood v. American Airlines, Inc.*, 107 F.3d 1565, 1572, 41 USPQ2d 1961, 1966 (Fed. Cir. 1997). Possession may also be shown in a variety of ways including description of an actual reduction to practice, or by showing that the invention was "ready for patenting" such as by the disclosure of drawings or

Art Unit: 1633

structural chemical formulas that show that the invention was complete, or by describing distinguishing identifying characteristics sufficient to show that the applicant was in possession of the claimed invention. See, e.g., *Pfaff v. Wells Elecs., Inc.*, 525 U.S. 55, 68, 119 S.Ct. 304, 312, 48 USPQ2d 1641, 1647 (1998); *Regents of the University of California v. Eli Lilly*, 119 F.3d 1559, 1568, 43 USPQ2d 1398, 1406 (Fed. Cir. 1997); *Amgen, Inc. v. Chugai Pharmaceutical*, 927 F.2d 1200, 1206, 18 USPQ2d 1016, 1021 (Fed. Cir. 1991) (one must define a compound by "whatever characteristics sufficiently distinguish it"). The applicant has not provided any description or reduction to practice of rodents resistance to collagen-induced arthritis other than an F1 C57Bl/6 X SJL mouse. Based on the applicant's specification, the skilled artisan cannot envision the detailed chemical structure of the genus of rodents encompassed by the claims. Therefore, conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. See *Fiers v. Revel*, 25 USPQ2d 1602 at 1606 (CAFC 1993) and *Amgen Inc. V. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016. Thus, for the reasons outlined above, claims 1-3, 5, and 8-12 do not meet the requirements for written description under 35 U.S.C. 112, first paragraph.

The rejection of pending claims 1-3, 5 and 7-12 under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement, is maintained in modified form. Applicant's amendments to the claims and arguments have been fully considered but have not been found persuasive in overcoming the rejection for reasons of record as discussed in detail below.

Art Unit: 1633

It is noted that based on the claim amendments and arguments, previously raised issues relating to post-partum gene transfer and the practice of the invention in Fcγ receptor knock-out mice by the previous examiner have been withdrawn.

The applicant has amended the claims to recite a method for screening a compound that is able to suppress an aberrant immune activity selected from aberrant immune complex formation, aberrant immune complex clearance, and immune complex induced inflammation comprising administering a compound to be screened to a transgenic rodent generated by transgenically modifying an embryo from a strain that is resistant to collagen-induced arthritis, such that said rodent comprises and expresses a transgene for human FcγRIIA receptor, whereby the expression of FcγRIIA receptor renders the rodent susceptible to an autoimmune disease, and assessing the rodent to determine if the compound reduces aberrant immune activity, and a method for screening a compound that is able to suppress an autoimmune disease comprising administering a compound to be screened to a cell derived from a transgenic rodent generated by transgenically modifying an embryo from a strain that is resistant to collagen-induced arthritis, such that said rodent comprises and expresses a transgene for human FcγRIIA receptor, whereby the expression of FcγRIIA receptor renders the rodent susceptible to an autoimmune disease, and assessing the cell to determine if the compound reduces aberrant immune activity.

The claims, however, are still broad and read on a transgenic rodent that is susceptible to any autoimmune disease, the use of any strain of rodent that is resistant to collagen-induced arthritis to produce the transgenic rodent as claimed, and methods of screening compounds by assessing any type of cell derived from the transgenic rodent as claimed to determine if the

Art Unit: 1633

compound reduces aberrant immune activity. The specification as filed does not provide an enabling disclosure for the breadth of the claims as written.

The previous office action set forth that the specification does not provide sufficient guidance for making any species of transgenic non-human mammal as previously claimed. The applicant has amended the claims to recite “a transgenic rodent generated by transgenically modifying an embryo from a strain that is resistant to collagen-induced arthritis, such that said rodent comprises and expresses a transgene for human FcγRIIA receptor, whereby the expression of FcγRIIA receptor renders the rodent susceptible to an autoimmune disease”. The applicant argues that this amendment overcomes the previous rejection because the specification provides disclosure for transgenic mice and it was routine in the art at the time of filing to make other transgenic rodents, such as rats and rabbits. The applicant also argues that Bezdicek et al., attached as evidence with the reply, shows that human FcγRIIA receptor is active in non-mouse rodents. In response, the order Rodentia includes approximately 1500 species of mammals, including mice, rats, hamsters, beavers, porcupines, squirrels, prairie dogs, and marmots. Please note that rabbits are not rodents, they belong to the order Lagomorpha, see for instance <http://www.ucmp.berkeley.edu/mammal/rodentia/rodentia.html>. However, as noted in the rejection of the claims for lack of written description above, of all the numerous species of rodents, the specification only discloses a single rodent that is resistant to collagen-induced arthritis, an F1 C57Bl/6 X SJL mouse. The specification does not provide any disclosure relating to any other mouse or any other type of rodent that is in fact resistant to collagen-induced arthritis. Further, the specification does not provide any guidance for rodent species other than the F1 C57Bl/6 X SJL mouse that becomes susceptible to any autoimmune disease, including

collagen-induced arthritis, following transgenic expression of human FcγRIIA receptor. The applicant argues that it would be routine to screen various mouse strains to determine others that are resistant to collagen-induced arthritis. However, the order rodents comprises approximately 1500 species whose susceptibility to arthritis and in particular collagen-induced arthritis is unknown. As such, the experimentation required to test each species of rodents and further various strains of rodents within each species would be undue.

In addition, the previous office action provided scientific reasons supported by citations from the prior art to show that the skilled artisan would not have been able to predict the phenotype of a transgenic mammal *a priori*. In particular, the previous office action stated that the manufacture of transgenic animals with a given phenotype are sensitive to factors such as the integration site of the transgene, copy number as well as the genetic background of the animal used. This observation is supported by Houdebine et al., who states that “numerous experiments have shown that the level and specificity of expression of a gene construct used as a transgene cannot be easily predicted” {Houdebine et al. (2000) Transgenic Research 9:305-320; pg. 309, col. 2: The expression of transgenes}. Further, Houdebine et al. states that the potency of any transgene can only be estimated in transgenic animals and the level of expression of transgenes in mice is not predictive of their levels in other animals (pg. 310, col. 1, pgph 2). Finally, Houdebine et al. states that another well known problem with transgenesis is leaky expression of the transgene in various tissues in which the utilized promoter is not expected to work because of ectopic expression due to a position effect (pg. 310, col.1, pgph 3). See also Kolb et al., who states that “the expression of foreign genes in transgenic animals is generally unpredictable as transgenes integrated at random after pro-nuclear injection into fertilized oocytes” because of

Art Unit: 1633

inhibition by neighboring chromatin {Kolb et al. (1999) Gene 227:21-31; Abstract}. Sigmund, C., concurs, reporting that variation in the genetic background contributes to the unpredictability of the resulting phenotypes of transgenic or gene-targeted animals {Sigmund, C., (2000) Arterioscler. Thromb. Vasc. Biol., p. 1425-1429}. “Animals containing the same exact genetic manipulation exhibit profoundly different phenotypes when present on diverse genetic backgrounds, demonstrating that genes unrelated, per se, to the ones being targeted can play a significant role in the observed phenotype” (e.g. abstract). Based on this evidence, the specification’s disclosure of a single transgenic mouse strain, a transgenic F1 C57Bl/6 X SJL mouse expressing human FcγRIIA receptor with the phenotype of reduced resistance to collagen-induced arthritis, does not provide sufficient enablement or guidance for making other mouse strains or rodent species with the same phenotype. Further, the evidence cited above makes it clear that the skilled artisan did not consider it “routine” to make a transgenic mammal, or even a rodent, with a particular phenotype.

Regarding the teachings of Bezdicek et al, it is noted that while Bezdicek et al. demonstrates that the expression of human FcγRIIA receptor in rat liver cells enhances liver uptake of opsonized red blood cells, Bezdicek et al. does not teach that expression of human FcγRIIA receptor in the liver results in reduced resistance to collagen-induced arthritis, or increased susceptibility to any autoimmune disease. Further, Bezdicek et al. is non-analogous art as Bezdicek et al. utilized adenoviral gene transfer to a specific adult organ, and does not teach or suggest making or using a transgenic rat. Thus, the teachings of Bezdicek et al. does not overcome the teachings of Houdebine et al., Kolb et al, and Sigmund cited above.

The following issues also lack enabling support in the specification as filed. As noted above, the claims read on a transgenic rodent in which the expression of FcγRIIA receptor renders the rodent susceptible to an autoimmune disease. There are numerous autoimmune diseases which variously effect different types of cells active in effecting immune responses, including T cells, B cells, eosinophils, dendritic cells, macrophages, NK cells etc. The specification does not provide an enabling disclosure for the production of a transgenic rodent in which the expression of FcγRIIA receptor renders the rodent susceptible to any autoimmune disease. As discussed in detail above and in the previous office action, the specification only provides guidance for the transgenic mice originally reported by McKenzie et al. The specification demonstrates that these mice have increased susceptibility to collagen-induced arthritis. However, collagen-induced arthritis is not analogous to other autoimmune diseases such as diabetes, lupus, or multiple sclerosis. The specification provides no evidence, nor does the prior art support any role for the FcγRIIA receptor in the pathogenesis of diabetes, lupus, or various other autoimmune diseases. McKenzie et al. only shows that the FcγRIIA receptor can affect platelet activity. Bezdicek et al. only shows that the FcγRIIA receptor can affect the uptake of opsonized antigen by liver cells. Thus, nothing in the specification or art provides any guidance or support for generating a phenotype of enhanced susceptibility to any autoimmune diseases, such as diabetes, lupus, or multiple sclerosis, other than collagen-induced arthritis in a transgenic F1 C57Bl/6 X SJL mouse expressing human FcγRIIA receptor or in any to other transgenic rodent expressing human FcγRIIA receptor. Since the specification does not provide an enabling disclosure for any transgenic rodent susceptible to any autoimmune disease as claimed, the specification further fails to provide sufficient enablement for screening any

Art Unit: 1633

compound capable of suppressing any autoimmune disease or aberrant immune activity as claimed.

In addition, claim 3 is drawn to a method of screening by administering the compound to a cell derived the transgenic rodent. The claim does not limit that the type of transgenic cell to be used in the screening method. The issues in regards to lack of enablement for any transgenic rodent as claimed have been addressed in detail above. However, even in regards to a transgenic F1 C57Bl/6 X SJL mouse expressing human FcγRIIA receptor, the specification fails to provide an enabling disclosure for using any cell derived from an F1 C57Bl/6 X SJL mouse expressing human FcγRIIA receptor to identify a compound capable of suppressing any autoimmune disease. The mice disclosed in the specification are mice made and reported by McKenzie et al., as discussed in the previous office actions. McKenzie et al., however, clearly teaches that human FcγRIIA receptor was only expressed in certain cell types, specifically marrow megakaryocytes and leukocytes, macrophages, and platelets (McKenzie et al., page 4315). McKenzie et al. reported that human FcγRIIA receptor expression was not found in lymphocytes. Since McKenzie et al. teaches that expression of human FcγRIIA receptor is limited to certain specific cell types in the transgenic F1 C57Bl/6 X SJL mouse, the skilled artisan would not have predicted success in using any other type of cell in the screening assays.

Thus, applicant's amendments, arguments, and evidence, do not overcome the rejection of record. Based on the specification as filed, the specification is only enabling for a method of screening a compound that is able to suppress an aberrant immune activity associated with collagen-induced arthritis comprising administering said compound to a transgenic mouse generated by transgenically modifying an embryo from an F1 C57Bl/6 X SJL mouse strain that

is resistant to collagen-induced arthritis, such that said mouse comprises and expresses a transgene for human FcγRIIA receptor, whereby the expression of FcγRIIA receptor renders the mouse susceptible to collagen-induced arthritis, and assessing the mouse to determine if the compound reduces said aberrant immune activity.

Claim Rejections - 35 USC § 102

The rejection of claims 1, 2, and 7-9 rejected 35 U.S.C. 102(b) as being anticipated by McKenzie et al. (1999) J. Immunol. 162: 4311-4318 is withdrawn in view of applicant's amendments and arguments.

However, new grounds of rejection of the claims under 35 U.S.C. 103(a) apply, see below.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out

Art Unit: 1633

the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1-2, 5, and 7-9 are newly rejected under 35 U.S.C. 103(a) as being unpatentable over McKenzie et al. (1999) J. Immunol. 162: 4311-4318. McKenzie et al. teaches a human FcγRIIA receptor transgenic non-human mouse on a C7BL/6 X SJL background (McKenzie et al., abstract; pg. 4312, Materials and Methods). McKenzie et al. describes three different mouse strains that were genetically modified to express the human FcγRIIA receptor (pg. 4316, Figure 6). McKenzie et al. describes the characterization of two mouse strains that were transgenic for human FcγRIIA receptor, on a wild type background (pg. 4316, Figure 6). Further, McKenzie et al. describes the characterization of a mouse strain that was transgenic for human FcγRIIA receptor, on an endogenous Fcγ knockout background (pg. 4316, Figure 6). It is noted that the exemplar human FcγRIIA receptor transgenic mouse disclosed in the instant application is one of the human FcγRIIA receptor transgenic mouse made by McKenzie et al. (Specification, pg. 12, lines 30-32; pg. 18, lines 15-20). This mouse inherently has decreased resistance to collagen-induced arthritis in comparison to control animals (Specification, pg. 27, Example 7). McKenzie et al. further teaches that the mice can be used as a model of autoimmune thrombocytopenia, wherein thrombocytopenia is assessed in terms of platelet counts (pg. 4313, co. 2; pg. 4316, col. 1, Figures 5 and 6). McKenzie et al. also provides specific motivation to use the FcγRIIA receptor transgenic mouse to screen therapeutic modalities (pg. 4317, col. 2). In particular, McKenzie et al. provides motivation to test therapies that diminish Ab effector mechanisms in

Art Unit: 1633

human immune thrombocytopenia directed at the expression or function of the FcγRIIA receptor (pg. 4317, col. 1). McKenzie et al. also teaches several compounds with the ability to suppress aberrant immune complex formation, clearance, and inflammation, including glucocorticoids and anti-D Ig (pg. 4311, col. 2) . Thus, in view of the motivation to test therapeutic modalities in the FcγRIIA receptor transgenic mouse provided by McKenzie et al., it would have been *prima facie* obvious to the skilled artisan at the time of filing to test compounds such as glucocorticoids and anti-IgD for their ability to suppress aberrant immune complex formation, clearance, and inflammation by administering them to the FcγRIIA receptor transgenic mouse and to assess the mouse for reduced aberrant immune activity with a reasonable expectation of success.

No Claims allowed.

Any inquiry concerning this communication from the examiner should be directed to Anne Marie S. Wehbé, Ph.D., whose telephone number is (571) 272-0737. If the examiner is not available, the examiner's supervisor, Dave Nguyen, can be reached at (571) 272-0731. For all official communications, **the new technology center fax number is (571) 273-8300**. Please note that all official communications and responses sent by fax must be directed to the technology center fax number. For informal, non-official communications only, the examiner's direct fax number is (571) 273-0737. For any inquiry of a general nature, please call (571) 272-0547.

The applicant can also consult the USPTO's Patent Application Information Retrieval system (PAIR) on the internet for patent application status and history information, and for electronic images of applications. For questions or problems related to PAIR, please call the

Application/Control Number: 10/517,251

Page 15

Art Unit: 1633

USPTO Patent Electronic Business Center (Patent EBC) toll free at 1-866-217-9197.

Representatives are available daily from 6am to midnight (EST). When calling please have your application serial number or patent number available. For all other customer support, please call the USPTO call center (UCC) at 1-800-786-9199.

Dr. A.M.S. Wehbé

ANNE M. WEHBE' PH.D
PRIMARY EXAMINER

A handwritten signature in black ink, appearing to read 'A.M.S. Wehbe', with a long horizontal line extending to the right.